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Session: Coinfections in HIV
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Time: 15:45-17:45
Room: Ballroom B

The role of coinfections in HIV epidemic trajectory and positive prevention

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Objectives: Previous studies have found that recurrent or persistent co-infections may increase HIV viral load (VL) in plasma and genital secretions. This elevation in HIV VL may increase the risk of HIV transmission, thus increasing HIV incidence. We evaluated the association between malaria, herpes simplex type 2 (HSV-2) and tuberculosis (TB) co-infections and their treatment on HIV VL.

Design: Systematic review and meta-analysis of the association of malaria, HSV-2 and TB co-infections and their treatment on HIV VL.

Methods: PubMed and Embase databases were searched to February 10th 2010 for studies in adults that reported HIV plasma and/or genital VL by co-infection status or treatment. Studies that adjusted for CD4 count or time since infection were included. Meta-analyses were conducted using random-effects models.

Results: Forty-five eligible articles were identified (6 malaria, 20 HSV-2 and 19 tuberculosis). There was strong evidence of increased HIV VL with acute malaria (0.67 log10 copies/mL, 95% CI: 0.15, 1.19) and decreased VL following treatment (-0.37 log10 copies/mL, 95% CI: -0.70, -0.04). HSV-2 infection was also associated with increased HIV VL (0.18 log10 copies/mL, 95% CI: 0.01, 0.34), which decreased with HSV suppressive therapy (-0.28 log10 copies/mL, 95% CI: -0.36, -0.19). Active tuberculosis was associated with increased HIV VL (log10 copies/mL 0.40, 95% CI: 0.13-0.67), but there was no association between tuberculosis treatment and VL reduction (log10 copies/mL -0.02, 95% CI -0.19, 0.15).

Conclusions: Co-infections may increase HIV VL in populations where they are prevalent, thereby facilitating HIV transmission. Treatment or co-infection prophylaxis could reverse the effect of co-infections. However, to limit HIV trajectory and optimize positive prevention for HIV-infected individuals pre-ART, we must better understand the mechanisms responsible for augmented VL and the magnitude of VL reduction required, and retune treatment regimens accordingly.

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Hepatitis B coinfection: Implication for management

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Background: Coinfection with human immunodeficiency virus (HIV) and hepatitis B (HBV) is a growing public concern, especially in Asia and Africa where both HIV and HBV are epidemic. The progression of chronic HBV to cirrhosis, end-stage liver disease and hepatocellular carcinoma is more rapid in HIV-infected persons than in persons with chronic HBV alone. Conversely, chronic HBV does not substantially alter the HIV disease progression and does not influence on CD4/HIV RNA response to ART. However, ARV related hepatotoxicity, hepatic flare due to immune recovery syndrome after initiating ART, lamivudine resistant, or HBV drug discontinuation can be associated with elevation in transaminases. First line treatment for chronic HBV mono infection is pegylated Interferon, tenofovir or entecavir. Since, lamivudine resistant is observed in 20-25% per year, therefore, lamivudine/emtricitabine should not be used as a mono HBV drug. Due to faster liver disease progression in HIV-HBV co-infection, ARV treatment should be initiated early. Combination of tenofovir plus emtricitabine or lamivudne as NRTI backbone is recommended for all HIV-HBV infected individuals in need for ART treatment. If HBV need treatment but HIV treatment is not indicated (CD4> 500 cells/mm3), HBV drug without HIV activity ie1) pegylated interferon for selected population (low HBV DNA, genotype A, absent cirrhosis); 2) telbivudine/adefovir or 3) early ART is suggested. For HIV-HBV patients with renal problem and tenofovir could not be safety used, there is currently no solid evidence to suggest the best strategy. The alternative option would be 1)entecavir in addition to a fully suppressive ART regimen, 2) pegylated interferon monotherapy, 3) adefovir plus emtricitabine or lamivudine, or 4) telbivudine in addition to a fully suppressive ART regimen. If ART needs to be modified due to HIV virological failure and patient has adequate HBV suppression, tenofovir and/or emtricitabine (or lamivudine) should be continued for HBV treatment in addition to other active ART regimen. Finally, primary prevention for HBV infection and screening for hepatocellular carcinoma is warranted.

Conclusion: This presentation will focus on the important of HBV and treatment strategy in HIV-HBV infected individual. In addition, HBV vaccine and primary prevention for hepatocellular carcinoma are also addressed.

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